Exhibit 23

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Expert Witness Report: Gary McElhiney

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II. Personal Background/Qualifications (change this entire section to 1st person)

I have been a practicing neurologist for over 30 years.

After completing a PhD in Neuroscience in the Department of Pathology at Northwestern University Medical Center, I entered medical school at Albert Einstein College of Medicine, completing my MD degree in 1989. I then pursued Neurology, with a residency at Columbia University in New York City where I was elected chief resident and completed my residency in 1993. Upon completion of my residency, I accepted a position as an Assistant Professor of Neurology at the University of Rochester. I have remained at the University of Rochester, working up through the academic ranks to become a full Professor in 2008. I became board certified by the American Board of Psychiatry and Neurology in 1994 and have remained board certified since.

I have remained clinically active over the last 31 years, practicing general neurology as well as subspecialty practices in peripheral nerve disorders and movement disorders. I

Contaminated Water at Camp Lejeune - TCE and PCE Exposure[1]

In my opinion, Mr. McElhiney had substantial exposure to TCE and PCE from his time at Camp Lejeune through the date of the official exposure period (December 31, 1987). This exposure was substantially more than nominal. He lived on and off base for 13 years, but even when living off base had continued to be exposed from showers and drinking during on-base activities.

TCE and PCE have been long known to cause health risks to human such that the US Environmental Protection Agent has recently banned their use. These two chemicals are recognized contaminants in the water at Camp Lejeune (*ATSDR 2017*). Exposures at Camp Lejeune have been shown to be a risk factor for the development of Parkinson's disease (*Goldman et al JAMA Neurol 2023; 80:673-681*)

There is ample evidence that neurotoxins are sufficient to cause neurodegenerative diseases such as Parkinson's disease *(Shaw and Hoglinger, Neuromolecular Med 2008; 10:8016-8)*. TCE is clearly one of those neurotoxins *(Goldman S. Ann Rev Pharmacol Toxicol 2014; 54:141-64)*. Epidemiological evidence for long-term TCE exposure being a risk factor for the development of Parkinson's disease has been increasing since it became apparent in 2008 *(Gash DM, et al. 2008; Ann Neurol 63:184-92)*. Strong evidence for this includes increased risk associated with TCE exposure in biological twins discordant for Parkinson's disease *(Goldman S, et al. Ann Neurol 71: 776-84)*. Twin studies are used to minimize the contribution of genetic factors as well as early life exposure factors.

TCE and PCE are structurally similar. While there are fewer existing data regarding PCE, there is no significanct difference between the two chemicals to suspect that PCE would not be equally toxic (*Cannon, General Causation report)*. It is notable that absorption of TCE occurs through skin absorption, gut absorption, and inhalation. This is an important consideration in the cases of Camp LeJeune as residents there both drank and showered in the water, resulting in exposure to all modes of entry into the body.

Given the weight of the evidence, exposure to the contaminated water at Camp Lejeune, is clearly an appropriate factor to consider when conducting a causality assessment using a differential diagnosis for Parkinson's disease.

Mr. McElhiney was exposed to the contaminated water at Camp Lejeune. Mr. McElhiney was stationed at Camp Lejeune for significant periods of time between June, 1972 and October, 1988. ATSDR Assessment (*Jan 13, 2017*), "A marine in training at Camp Lejeune

[1] The causal relationship between the water at Camp Lejeune (most notably TCE and PCE) and Parkinson's disease is set out in detail in the general causation reports of Drs. Boehme, De Miranda, Miller, Cannon, Costa, Freeman, and Bird). As noted above, I have reviewed, considered, and understand these reports as to the general issues concerning the causal relationship between TCE, PCE and Parkinson's disease. Although I have deferred to their specific expertise, I have reviewed these reports and I agree that these toxins are at least as likely as not to cause Parkinson's disease.

known mechanism in which this toxin causes the disease. [redacted] By the time Parkinson's disease is clinically manifested, it is estimated that the underlying death of dopaminergic neurons has been progressing over the prior 15-20 years. That indeed may be an underestimate as recent studies of Parkinson's disease prodrome symptom of REM behavior disorder can precede the diagnosis by even more years. On top of that, TCE induced cell death is not instantaneous, and therefore one would need to add on the time it takes for the toxin to *start* to cause the death of neurons.

Assessing the evidence addressed in this report, and based upon my education, training, and experience as a neurologist and specialist in movement disorders, it is my opinion that TCE is at least as likely as not a cause of Mr. McElhiney's Parkinson's disease. Although this diagnosis/etiology opinion can be based on clinical findings and judgment alone, it can also be supported by an application and consideration of the Bradford Hill criteria, not all of which need to be met, but individually:

1. Temporal relationship: In my opinion, as above, given the known decades-long prodrome for the development of clinical Parkinson's disease, the latency between the Mr. McElhiney's exposure to contaminated water in Camp Lejeune and the development of his Parkinson's disease meets this criterion.
2. Consistent positive associations: There is consistency between epidemiologic studies of exposure to TCE and the development of Parkinson's disease in other settings outside of the Camp Lejeune exposure. Another study in humans also showed an increased risk of Parkinson's disease in the general population exposed to TCE (*Exposure to industrial Solvent Linked to 24% Higher PD Risk- Medscape- April 26, 2024*). In my opinion, this criterion is met.
3. Magnitude of the effect estimate: It is more likely than not that Mr. McElhiney's estimated exposure to TCE exceeds those exposure levels linked to disease. (*Miller, General Causation report)*
4. Exposure-response relationship
5. Biological plausibility: TCE is a known neurotoxin that causes several dysfunctions in neurons that lead to the development of parkinsonism.
6. Coherence: there is clear coherence between the disease produced in laboratory animals and human Parkinson's disease. An animal model using this specific neurotoxin reproduces of all the key features that are seen in human Parkinson's disease (*Liu M et al J Neurochem 2010, 112:773* ; *De Miranda, General Causation report*).

7. Strength: the Goldman study comparing Marines at Camp Lejeune with those from Camp Pendelton was a large study with a significant effect size

I considered the other two criteria of analogy and experimentation (human), but I did not feel the former applied in this situation, and the latter is clearly not feasible.

Other Factors Considered

In conducting my differential diagnosis, I have also considered other possible causes of Mr. McElhiney's Parkinson's disease. The potential causes I considered are necessarily limited to the possible causes to which Mr. McElhiney was exposed given his work and life history. [redacted]